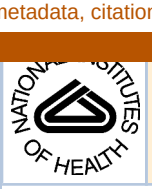

Published in final edited form as:

Nutr Res. 2008 September ; 28(9): 565–576. doi:10.1016/j.nutres.2008.06.005.

Correlates of Antioxidant Nutrients and Oxidative DNA Damage Differ by Race in a Cross-Sectional Study of Healthy African American and White Adults

Joanne L. Watters[1], **Jessie A. Satia**[2,3,4,5], and **Lawrence L. Kupper**[6]

1*Cancer Prevention Fellowship Program, Office of Preventive Oncology, Division of Cancer Prevention, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20852*

2*Department of Nutrition, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599*

3*Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599*

4*Lineberger Comprehensive Cancer Center, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599*

5*Center for Gastrointestinal Biology and Disease, Division of Digestive Diseases and Nutrition, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599*

6*Department of Biostatistics, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599*

Abstract

Although antioxidant nutrients and oxidative DNA damage have been associated with carcinogenesis, few studies have investigated the factors that influence antioxidant intake and oxidative DNA damage in racially diverse populations. Demographic, behavioral, and diet-related psychosocial correlates of plasma antioxidant (carotenoids, vitamin C, and vitamin E) concentrations and oxidative DNA damage were examined using data from a cross-sectional study of 147 generally healthy, non-smoking African American and White adults in North Carolina, age 20 to 45 years. All participants completed self-administered demographic, diet, and health questionnaires and provided semi-fasting (≥ 6 hours) blood samples. Multivariate regression analyses were computed separately for each race to determine associations between the potential correlates with plasma antioxidant concentrations and oxidative DNA damage, separately. Our findings suggest appreciable differences by race. Only a few factors (age, supplement use, and several psychosocial factors) were associated with antioxidant concentrations in African Americans, whereas these and additional factors, including physical activity, waist circumference, and passive smoke exposure, were associated with antioxidant concentrations in Whites. For oxidative DNA damage, passive smoke exposure was significantly associated with oxidative DNA damage in African Americans, and age and alcohol were significant in Whites. In addition, the regression models generally explained more of the variance in plasma antioxidant concentrations and oxidative DNA damage in Whites than in African Americans. Considering the salient correlates differed by race, this work has important implications for the design and implementation of future research studies investigating antioxidant nutrients and/or oxidative stress, especially those in racially diverse populations.

Corresponding Author: Joanne L. Watters, PhD, MPH, Cancer Prevention Fellow, NIH/NCI, 6130 Executive Blvd, Suite T-41 (EPS), Bethesda, Maryland 20852, Tel: 301-435-2758, Fax: 301-480-2669, Email: wattersj@mail.nih.gov.

Keywords

Oxidative Stress; carotenoids; vitamin C; vitamin E; African Americans; Humans

1. Introduction

There is considerable interest in the roles of antioxidant nutrients and oxidative DNA damage in carcinogenesis. Antioxidants are substances within many foods that decrease the adverse effects of reactive oxygen species (ROS) on normal physiological functions [1]. High ROS levels can lead to oxidative stress, in which the imbalance of radical-generating agent concentrations exceeds the body's defense mechanisms [2,3]. Excess oxidative stress can lead to oxidative damage of DNA, which is thought to be a significant contributor to the age-related development of cancer [4–6]. Although most observational studies provide support for a protective association between high dietary intakes and/or supplemental doses of antioxidant vitamins with cancer risk [7], two randomized trials found elevated lung cancer risk with high-dose supplementation in high-risk populations [8,9].

Given the associations of antioxidants and oxidative DNA damage with cancer, identifying factors that may influence these levels is important for several reasons. First, it will provide information on key factors that need to be included in the design of research studies and identify potential confounders that are necessary for appropriate statistical analyses and interpretation of study results. Second, this information may identify mediating variables for cancer prevention initiatives. Studies that have investigated factors related to antioxidant and oxidative DNA damage levels have most often focused on demographic characteristics, such as age and gender, or behavioral factors, such as smoking, diet, and alcohol use [10–14]. Considering that fruit and vegetable intake is a strong determinant of blood antioxidant concentrations [16], it is critical that psychosocial factors also be examined as they have been found to explain a moderate amount of variation in fruit and vegetable consumption [17].

There is strong evidence that antioxidant and oxidative DNA damage levels differ between African Americans and Whites. Previous studies have shown lower serum concentrations of most antioxidants [18–21] and also lower oxidative DNA damage levels [21–23] in African Americans than Whites. Moreover, African Americans are at disproportionately higher risk for many oxidative stress-related medical conditions, such as cancer [24]. To date, investigations of the various participant characteristics that influence antioxidant status and oxidative DNA damage have been most often conducted in largely White populations; however, further investigation of these issues is warranted, particularly in racially diverse populations.

This study examined potential racial differences in demographic, behavioral, and diet-related psychosocial correlates of plasma carotenoid, vitamin C, and vitamin E concentrations and oxidative DNA damage in a sample of healthy African American and White adults. In a previous study, we found that African Americans had lower plasma concentrations of antioxidant nutrients and also had lower oxidative DNA damage than White adults after adjustment for known risk factors [21]. However, we did not explore the factors contributing to the each of the levels observed separately by race. Considering the differing antioxidant concentrations and oxidative DNA damage levels by race, identification of factors has important implications for the design and implementation of research studies investigating antioxidant nutrients and/or oxidative stress.

2. Methods and materials

2.1. Study population

Data are from the DIet, Supplements, and Health (DISH) Study, which enrolled 168 generally healthy African American and White adults (approximately equal by race and gender) from the Research Triangle Area of North Carolina between March and December 2005. Details of this study have been published elsewhere [21]. Briefly, eligible participants were recruited via flyers displayed in public venues and were 20 to 45 years of age, non-smokers, fluent in written and spoken English, and had no personal history of cancer, diabetes, heart disease, or Alzheimer's disease. Because obesity is associated with oxidative stress, participants with a body mass index (BMI) ≥ 30 were ineligible. Data were excluded for 8 participants with measured BMIs ≥ 30 and 9 participants with levels of cotinine, a metabolite of nicotine, which were consistent with active smoking (≥ 15 ng/mL); 147 participants remained (70 African American, 77 White). This study was approved by the University of North Carolina (UNC)'s Institutional Review Board and written informed consent was obtained from all participants.

2.2. Data collection

Participants had height, weight, and waist circumference measured and provided semi-fasting (≥ 6 hours) blood samples at the UNC's General Clinical Research Center. At home, participants completed a 12-page demographic, health, and antioxidant questionnaire, which was adapted from previous research [25–27] and contained questions about physical activity, attitudes and beliefs regarding diet, smoking, alcohol use, demographics, and supplement use. Blood samples were analyzed for plasma antioxidant nutrients, cholesterol, oxidative DNA damage, hemoglobin A1c (to confirm self-reported absence of diabetes), and serum cotinine (to validate self-reported smoking status). Although both self-reported dietary intakes and plasma concentrations were available, plasma concentrations were used in these analyses to obviate the limitations of self-reported data [28].

2.3. Demographic, Behavioral, and Psychosocial Characteristics

All demographic, behavioral, and psychosocial characteristics were assessed using information from the demographic, health, and antioxidant questionnaire, including sex, age, education (some college or less, college graduate, or advanced degree), and income (<$20,000; $20–39,000; $40–79,000; $80,000+). BMI, calculated in kg/m^2 using height and weight measurements, was categorized as normal (18.5 to 24.9) and overweight (25.0 to 29.9) [29]. Three repeated waist circumference measurements were averaged and then tertiles were computed separately by sex.

Usual physical activity was captured as frequency per week (<2, 3–4, 5+ times/week). A single item question was used for usual alcohol consumption (none, <1/week, 1–6/week, 1–2/day, 2+/day). Passive smoke exposure was assessed by asking whether *anyone in the household smokes now* (yes/no). Supplement use was queried in a closed-ended format that quantified frequency and dose of multivitamins and herbal supplements. Specifically, for multivitamin use, participants selected from a list of common multivitamins or wrote in their brand if it was not listed, and indicated the usual frequency of use (number of days per week). Next, they reported whether they took any herbal or single nutrient supplements and if so, the frequency and usual dose (amount per day). For these analyses, participants were categorized as "non-users" and "users" of multivitamin and herbal supplements (treated separately), if they reported any use *in the past month*.

Participants were asked whether they believe a diet and cancer relationship exists, and if so, whether the relationship is strong, moderate, or weak; whether they believe antioxidants are good for health; how many servings of fruits and vegetables one *should* eat each day for good

health 5+, 3–4, 1–2, not sure/don't know); how important it is for them personally to eat a diet high in fruits and vegetables (very, somewhat, or not important); and self-efficacy, which was assessed as respondents' confidence (very, somewhat, or not confident) in their ability to eat more fruits and vegetables. Participants were also asked whether they felt they could afford healthy foods, such as fruits and vegetables (yes, no, sometimes).

2.4. Biological Samples

Semi-fasting (≥ 6 hours) blood samples that were protected from heat and light were analyzed for plasma concentrations of carotenoids, tocopherols, and vitamin C using high performance liquid chromatography (HPLC) with multiwavelength photodiode-array absorbance detection by Craft Technologies Inc. (Wilson, NC) [30]. The aliquot of plasma designated for ascorbic acid assessment was preserved with a 6% weight/volume metaphosphoric acid (MPA) solution added in 1:4 plasma to MPA ratio to stabilize vitamin C. Plasma cholesterol was measured by enzymatic/colorimetric analyses using adaptations of commercially available kits Kit No. 401-25P, Sigma-Aldrich, St. Louis, MO). Multiple levels of quality control samples were included and 10% blinded duplicates were included in each batch. All samples were stored at −80°C and analyzed within one year, well within guidelines for storage stability [31].

Oxidative DNA damage was assessed using the alkaline comet assay, a widely used method for measuring DNA strand breaks at the level of a single cell [32,33]. The comet assay used here was a slightly modified version in which formamidopyrimidine DNA glycosylase (FPG) (provided by Dr. A.R. Collins, Oslo, Norway) was added to convert oxidized purines into strand breaks [34]. Peripheral whole blood lymphocytes were washed in PBS, counted using a hemacytometer, and cryopreserved in 1 ml RPMI-1640 + 15% BSA+ 10% DMSO. All samples were processed within 2 hours of collection and stored at –80°C until assays were performed, as recommended by the European Standards Committee on Oxidative DNA Damage (ESCODD) [34]. Comet tail length (the distance of DNA migration from the body of the nuclear core) was visualized by using a fluorescence microscope and SCION IMAGE software [35]. The comet tail moment (defined as the integrated density in the comet tail multiplied by the distance from the center of the nucleus to the center of mass of the tail) was calculated by using the NIHIMAGEANALYSISMACRO language software [35]. Multiple levels of quality control samples (e.g., 10% blinded duplicate samples) were included in each batch and all assays were performed by the University of North Carolina Clinical Nutrition Research Unit.

2.5. Statistical analysis

Data analyses were performed using Stata (version SE 8.2, STATACorp, College Station, TX). Descriptive statistics were calculated for all variables; missing data were excluded from analyses. For each study population characteristic, chi-square tests were used to test for equality by race. Mean plasma antioxidant nutrients concentrations and oxidative DNA damage levels were reported separately by race for each factor and potential racial differences were evaluated using analysis of variance and tests for linear trend, where appropriate. Log transformations were applied to the antioxidant and oxidative DNA damage right-skewed distributions to help meet normality assumptions. Plasma cholesterol was included in all analyses evaluating fat-soluble antioxidants, as it affects bioavailability [16]. Forward stepwise regression analyses, with a variable inclusion criterion of 0.05, were computed separately for each race to determine associations between the demographic, behavioral, and diet-related psychosocial correlates with plasma antioxidant concentrations [36]. Because there were fewer relevant demographic and behavioral correlates suggested in the literature, multiple linear regression models were used instead of stepwise regressions to examine these correlates with oxidative DNA damage [36]. Statistical tests were two-sided and p values ≤0.05 identified statistically significant associations.

3. Results

The distributions of demographic and lifestyle characteristics, stratified by race (n=147) are given in Table 1. The mean age of African American participants was 30.6 years (8.0 SD) and 56% were female; in comparison, the mean age for Whites was 32.4 years (7.8 SD) and 53% were female. African Americans had statistically significantly lower formal educational levels, physical activity, and alcohol consumption than Whites and were also more likely to be overweight.

Table 2 gives the mean plasma antioxidant concentrations and oxidative DNA damage levels for each of the demographic correlates examined. In general, age was positively associated with plasma concentrations of antioxidants in Whites, but not African Americans. A healthy BMI (<25 kg/m^2) tended to be associated with higher concentrations of most antioxidants, with statistically significant associations for vitamin C in African Americans and lutein+zeaxanthin in Whites. Waist circumference was positively associated with vitamin E, yet inversely associated with vitamin C. Participants who reported higher incomes or education levels generally had higher antioxidants concentrations. There was a significant positive association for oxidative DNA damage with waist circumference in African Americans.

Mean plasma antioxidant concentrations and oxidative DNA damage levels for the behavioral correlates are presented in Table 3. Physical activity was generally positively associated with plasma antioxidant concentrations, with statistically significant associations for β-carotene and lutein+zeaxanthin in Whites. All antioxidant plasma concentrations, except lycopene, were higher in multivitamin and herbal supplement users of both races. “Living with a smoker” was associated with lower oxidative DNA damage in African Americans (p=0.01), whereas alcohol consumption was significantly inversely associated with oxidative DNA damage in Whites.

Mean plasma antioxidant concentrations for each psychosocial correlate are presented in Table 4. Those who *believe there is a “strong” relationship between diet and cancer risk* had higher concentrations of most antioxidants with statistically significant associations seen for β-carotene and lutein+zeaxanthin in African Americans. Similarly, those who believe *antioxidants are good for health* generally had higher antioxidant concentrations. Those who had *knowledge of FV recommendations* had statistically significant higher lutein+zeaxanthin (both races) and vitamin E concentrations (Whites). The *importance of a diet high in FV* was positively associated with vitamin E concentrations in both races. Among Whites, those *able to afford healthy foods* had significant higher vitamin E and lutein+zeaxanthin concentrations.

Table 5 gives the results from the stepwise regression analyses (inclusion criteria ≤ 0.05) examining demographic, behavioral, and psychosocial correlates with antioxidant plasma concentrations, stratified by race. Vitamin C concentrations were approximately 30% higher in herbal supplement users of both races compared non-users, as well as African Americans who *believed antioxidants were good for health*. Age, multivitamin use, *belief in the importance of a diet high in fruits and vegetables*, and cholesterol accounted for 47% of variance in vitamin E among Whites, whereas 25% of variance in African Americans was explained by *belief in the importance of a diet high in fruits and vegetables* and cholesterol only. Whites who were at least 29 years of age, participated in physical activity 3x/week or more, and did not live with a smoker had approximately 50% higher plasma β-carotene concentrations than those who did not (R^2=0.44). For lutein+zeaxanthin, the final model included cholesterol and *belief in the diet and cancer link* (R^2=0.18) in African Americans; and cholesterol, herbal supplement use, and *belief in the importance of a diet high in fruits and vegetables* in Whites (R^2=0.21). Age was inversely associated with plasma lycopene concentrations in African Americans; cholesterol, waist circumference, and *belief in the diet and cancer link* were significantly correlated with lycopene in Whites. Plasma cholesterol was

forced into all models for fat-soluble nutrients, and explained 25% and 21% of the variability in Vitamin E in African Americans and Whites, respectively, about 10% for lycopene (both races), and < 5% for β-carotene and lutein+zeaxanthin in both races (data not shown).

Regression analyses examining variables that have been associated with oxidative DNA damage in the published literature are given in Table 6. These demographic and behavioral correlates explained 19% of variation in oxidative DNA damage in African Americans and 27% in Whites. For African Americans, passive smoke exposure was statistically significantly positively associated with oxidative DNA damage; those who lived with a smoker had 19% higher oxidative DNA damage levels than those with who did not. In Whites, oxidative DNA damage was significantly associated with age 38–45 years (15% higher than age 20–28 years) and consumption of ≥ 1 alcoholic drink per week (approximately 25% lower than non-drinkers).

4. Discussion

In this cross-sectional study of healthy African American and White adults in North Carolina, we examined demographic and behavioral correlates of plasma antioxidant concentrations and oxidative DNA damage. Our findings suggest appreciable differences in the salient correlates for both antioxidants and oxidative DNA damage by race. Furthermore, the regression models generally explained more of the variance in plasma antioxidant concentrations and oxidative DNA damage in Whites than in African Americans.

4.1 Demographic correlates and antioxidant concentrations

Generally, older age is positively associated with antioxidant concentrations [10,14]; however age was *inversely* associated with plasma lycopene in African Americans in the regression analysis. This inverse association may be an anomaly in this sample; however, it supports the need to examine potential confounders separately by race. Income was significantly positively correlated with concentrations of several antioxidants, which is likely due to a difference in fruit and vegetable intake, as national survey data indicate that intake is lower for those with lower incomes [37]. Although gender was not associated with any of the antioxidants in the stepwise regression models, there were statistically significantly higher mean vitamin C and lycopene concentrations in White men. There were not obvious trends by gender for vitamins C, E, and β-carotene, but it appeared that lutein+ zeaxanthin may be higher in women, whereas lycopene was higher in men of both races. Gender has been shown to be an important influence in serum antioxidants [10–12], as well intake of fruits and vegetables, which are naturally high in antioxidants [7,16].

Waist circumference was positively associated with lycopene and vitamin E, yet inversely associated with lutein+zeaxanthin and vitamin C. Similar findings were reported in a cross-sectional Swedish study in which waist circumference was positively associated with vitamin E, but inversely associated with β-carotene concentrations [38]. Two explanations were offered for this inverse association: 1) since β-carotene is stored in fat tissue, those with excess tissue would store more β-carotene and thus, have lower circulating plasma levels, or 2) obese persons may consume fewer FV, which are antioxidant-rich [38]. Given that those with a self-reported BMI >30 kg/m^2 were ineligible, the relationships seen here for waist circumference are especially notable and would likely be even more striking in samples with a wider range of anthropometric values. Although BMI was not found to affect antioxidant concentrations here, it is important to note that other studies have found lower antioxidant concentrations in obese participants [10,14].

4.2 Behavioral correlates and antioxidant concentrations

Physical activity frequency was associated with higher β-carotene concentrations in Whites, which is consistent with previous work that found higher serum antioxidant levels of carotenoids in those who were physically active in the past month using data from the National Health and Nutrition Examination Survey (NHANES) III, a cross-sectional, nationally-representative survey of adults 17 years and older [15]. Serum β-carotene was 53% lower for those living with a smoker in Whites. One might have expected a minimal effect of smoking considering that all participants were nonsmokers and only 6% lived with smokers; however this association supports other studies that have indicated smoking strongly influences antioxidant concentrations [10,16,38].

Plasma concentrations were consistently higher for all antioxidants, except lycopene, in multivitamin users compared to non-users. Vitamin E concentrations were 17% higher among White multivitamin users compared to non-users, which reflects the large contributions supplements make to total vitamin and mineral intakes [39]. It is somewhat surprising that there was not a similar association in African Americans, but this is likely due to the lower use of supplements in African Americans seen here (34% of African Americans vs. 50% of Whites reported taking supplements). Our findings are supported by results from the Multiethnic Cohort Study in which only 43% of African American participants reported supplement use compared to 57% of Whites [40]. When we examined the dietary supplement use patterns further by race, we found higher serum vitamin E levels in those who reported supplement use compared to those who did not for both races, as expected. However, there were far fewer African Americans reporting supplemental vitamin E use. For those reporting dietary supplement use, the frequency of use did not differ by race (45% of supplement-users of each race reported daily use). Overall trends were similar for multivitamin and herbal supplement users; vitamin C concentrations were approximately 30% higher in herbal supplement users of both races, which is not unexpected as many herbal supplements also contain vitamins and minerals. The most commonly reported supplements in this sample were ginseng, which naturally contains vitamin C [41], and glucosamine/chondritin, which can be packaged with vitamin C.

4.3 Diet-related psychosocial correlates and antioxidant concentrations

The most salient psychosocial factors based on the regression analyses appeared to be: *belief in the link between diet and cancer, belief in the importance of a diet high in FV,* and *belief that antioxidants are good for health.* These findings are in agreement with studies that have found that the individuals' knowledge, attitudes, and beliefs, such as believing in a diet and disease association, have statistically significantly higher fruit and/or vegetable intakes [42, 43]. Consistent with ideas from the Theory of Planned Behavior, those who value a behavior, such as healthy eating, are more likely to engage in that behavior [44]. Considering fruits and vegetables are good sources of many antioxidants, it logically follows the belief that a *diet high in fruits and vegetables is important* would be associated with higher blood antioxidant levels, as evident here. Serum vitamin E was about 10% higher in those who *believed a diet high in fruits and vegetables was important* for both races; interestingly, fruits and vegetables are better sources of the other antioxidants examined here. Perhaps that accounts for why this factor explains a greater amount of lutein +zeaxanthin (β coef=0.259) than vitamin E. In African Americans, *belief that antioxidants are good for health* was associated with statistically significant higher vitamin C in African Americans but not Whites, probably because there was little variation among Whites as most (87%) shared this belief. Surprisingly, *self-efficacy to eat a diet high in FV* was unrelated to serum antioxidants here. However, this is likely due to the small number of participants in this healthy population who reported low self-efficacy, defined as the extent to which one believes s/he can successfully perform a given behavior,

since self-efficacy has consistently been shown to positively influence healthy dietary behavior [17,42,43].

4.4 Demographic and behavioral correlates with oxidative DNA damage

Living with a smoker was significantly associated with oxidative DNA damage in African Americans, as were age and alcohol intake in Whites. Each model explained approximately 20% of the variance, suggesting there are salient factors that were either not included or adequately captured in these analyses. Our results in African Americans are in agreement with other studies showing that smoking is associated with elevated oxidative DNA damage levels [45,46]. Since our sample was restricted to non-smokers, we used whether *they lived with a smoker* as a proxy for passive smoke exposure. Interestingly, when mean oxidative DNA damage was compared by passive smoke exposure without adjusting for any covariates, damage was statistically significantly *lower* for African Americans living with a smoker. However, these results were confounded by age as all but one person who reported living with a smoker was in the youngest age category.

In Whites, those who reported consuming one or more drink per week had lower oxidative DNA damage than those who reported never consuming alcohol. A similar, although non-statistically significant, pattern was also reported in a study comparing oxidative stress measured as lipid peroxidation in healthy adults 19–78 years [47]. Heavier drinking has been associated with increased oxidative stress [48]; however, less than 1% of our sample reported consuming 2 or more drinks per day and thus, was not a concern here. Age was also associated with oxidative DNA damage in Whites, although the relationship was not linear, as the middle category of age (29 to 37 years) had the lowest oxidative DNA damage. It is possible that this sample was too young to see the effects of aging, as differences by age have usually been seen in persons over age 60 [11,49]. It is notable that the significant correlates of oxidative DNA damage we identified were not similar for African Americans and Whites, which emphasizes the importance of examining correlates of oxidative DNA damage separately by race.

4.5 Strengths, Limitations, and Conclusion

Our study has several strengths. The survey instrument was adapted from questionnaires that have been used in other studies [25–27]. Plasma concentrations of antioxidant nutrients were assessed using biomarkers, which are objective measures unaffected by many of the biases associated with self-reported dietary intake [28], and oxidative DNA damage was measured using a modified comet assay with FPG, which is considered to be an optimal measure for oxidative stress [50]. Finally, it is among the first to examine the correlates of plasma antioxidant concentrations and oxidative DNA damage in a sample with an adequate representation of African Americans.

We also acknowledge some limitations. First, self-reported data are subject to both random and systematic bias [16]. Second, the fact that our study population consisted of generally healthy volunteers may limit generalizability. Furthermore, the exclusion of current/former smoking, obese, older, or chronically-ill participants precluded our ability to evaluate these likely correlates of antioxidant concentrations and oxidative DNA damage. Third, the limited sample size may obscure some of the associations examined, especially for those variables with multiple responses stratified by race. Fourth, some measures designed to capture complex behaviors, e.g., physical activity, were measured using one or two self-reported items. Fifth, the psychosocial factors we examined are not a complete sampling of the possible psychosocial variables that could be studied in this context. Last, due to the cross-sectional nature of this study, no inferences can be made regarding causality.

In summary, we found that significant correlates of antioxidant concentrations and oxidative DNA damage differ for African Americans and Whites. Therefore, it is crucial to measure these items in future, larger studies so that potential important racial differences can be examined. Generally, the regression models explained more of the variance in plasma antioxidant concentrations and oxidative DNA damage in Whites than in African Americans, which may reflect the lack of existing information from studies with racially diverse populations. These results generally confirm other studies suggesting that demographic, behavioral, and psychosocial correlates influence plasma antioxidant concentrations. Age, alcohol intake, and smoking (passive and active exposure) should be examined in all investigations of oxidative DNA damage. Additional studies using similar methods but with larger demographically-diverse samples with wide variability in salient variables, such as age, race, BMI, and smoking exposure, are needed so that data can be stratified and analyzed with adequate statistical power.

LIST OF ABBREVIATIONS

BMI, Body Mass Index; DISH, DIet, Supplements, and Health Study; ESCODD, European Standards Committee on Oxidative DNA Damage; FPG, Formamidopyrimidine DNA Glycosylase; MPA, Metaphosphoric Acid; ROS, Reactive Oxygen Species.

Acknowledgments

This study was supported in part by the following grants: RO3 CA108276, K22 CA96556, T32 CA72319, P30ES010126, DK56350, and RR00046.

References

1. National Academy of Sciences. Dietary References Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Washington, DC: National Academy Press; 2000.
2. Sies, H. Oxidative stress: Oxidants and Antioxidants. San Diego, CA: Academic Press; 1991. Oxidative Stress: introduction.
3. Ames BN. DNA damage from micronutrient deficiencies is likely to be a major cause of cancer. Mutat Res 2001;475:7–20. [PubMed: 11295149]
4. Simic MG. DNA markers of oxidative processes in vivo: relevance to carcinogenesis and anticarcinogenesis. Cancer Res 1994;54:1918s–1923s. [PubMed: 8137312]
5. Collins AR, Dusinska M, Gedik CM, Stetina R. Oxidative damage to DNA: do we have a reliable biomarker? Environ Health Perspect 1996;104:465–469. [PubMed: 8781365]
6. Kang DH. Oxidative stress, DNA damage, and breast cancer. AACN Clin Issues 2002;13:540–549. [PubMed: 12473916]
7. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington DC: AICR; 2007.
8. Goodman GE, Thornquist MD, Balmes J, Cullen MR, Meyskens FL, Omenn GS, et al. The Beta-Carotene and Retinol Efficacy Trial: incidence of lung cancer and cardiovascular disease mortality during 6-year follow-up after stopping beta-carotene and retinol supplements. J Natl Cancer Inst 2004;96:1743–1750. [PubMed: 15572756]
9. Albanes D, Heinonen OP, Huttunen JK, Taylor PR, Virtamo J, Edwards BK, et al. Effects of alpha-tocopherol and beta-carotene supplements on cancer incidence in the Alpha-Tocopherol Beta-Carotene Cancer Prevention Study. Am J Clin Nutr 1995;62:1427S–1430S. [PubMed: 7495243]
10. Galan P, Viteri FE, Bertrais S, Czernichow S, Faure H, Arnaud J, et al. Serum concentrations of [beta]-carotene, vitamins C and E, zinc and selenium are influenced by sex, age, diet, smoking status, alcohol consumption and corpulence in a general French adult population. Eur J Clin Nutr 2005;59:1181–1190. [PubMed: 16034362]
11. Mendoza-Nunez VM, Sanchez-Rodriguez MA, Retana-Ugalde R, Vargas-Guadarrama LA, Altamirano-Lozano MA. Total antioxidant levels, gender, and age as risk factors for DNA damage

in lymphocytes of the elderly. Mechanisms of Ageing and Development 2001;122:835–847. [PubMed: 11337012]

12. Natarajan L, Flatt SW, Sun X, Gamst AC, Major JM, Rock CL, et al. Validity and Systematic Error in Measuring Carotenoid Consumption with Dietary Self-report Instruments. Am. J Epidemiol 2006;163:770–778. [PubMed: 16524958]
13. Talegawkar SA, Johnson EJ, Carithers T, Taylor HA Jr, Bogle ML, Tucker KL. Total alpha-tocopherol intakes are associated with serum alpha-tocopherol concentrations in African American adults. J Nutr 2007;10:2297–2303. [PubMed: 17885014]
14. Andersen LF, Jacobs DR Jr, Gross MD, Schreiner PJ, Dale Williams O, Lee DH. Longitudinal associations between body mass index and serum carotenoids: the CARDIA study. Br J Nutr 2006;2:358–365. [PubMed: 16469154]
15. Stimpson JP, Nash AC, Ju H, Eschbach K. Neighborhood Deprivation Is Associated with Lower Levels of Serum Carotenoids among Adults Participating in the Third National Health and Nutrition Examination Survey. J Am Diet Assoc 2007;107:1895–1902. [PubMed: 17964308]
16. Willett, WC. Nutritional epidemiology. New York, NY: Oxford University Press; 1998.
17. Van Duyn MA, Kristal AR, Dodd K, Campbell MK, Subar AF, Stables G, et al. Association of awareness, intrapersonal and interpersonal factors, and stage of dietary change with fruit and vegetable consumption: a national survey. Am J Health Promot 2001;16:69–78. [PubMed: 11727591]
18. Ford ES, Schleicher RL, Mokdad AH, Ajani UA, Liu S. Distribution of serum concentrations of alpha- and gamma-tocopherol in the US population. Am J Clin Nutr 2006;84:375–383. [PubMed: 16895886]
19. Ford ES. Variations in Serum Carotenoid Concentrations Among United States Adults by Ethnicity and Sex. Ethn Dis 2000;10:208–217. [PubMed: 10892827]
20. Satia-Abouta J, Galanko JA, Martin CF, Potter JD, Ammerman A, Sandler RS. Associations of Micronutrients with Colon Cancer Risk in African Americans and Whites: Results from the North Carolina Colon Cancer Study. Cancer Epidemiol Biomarkers Prev 2003;12:747–754. [PubMed: 12917206]
21. Watters JL, Satia JA, Schroeder JC, Switzer BR, Swenberg JA, Kupper LL. Association of antioxidant nutrients and oxidative DNA damage in healthy African Americans and White adults. Cancer Epidemiol Biomarkers Prev 2007;16:1428–1436. [PubMed: 17627008]
22. Huang HY, Helzlsouer KJ, Appel LJ. The Effects of Vitamin C and Vitamin E on Oxidative DNA Damage: Results from a Randomized Controlled Trial. Cancer Epidemiol Biomarkers Prev 2000;9:647–652. [PubMed: 10919732]
23. Toraason M, Butler MA, Ruder A, et al. Effect of perchloroethylene, smoking, and race on oxidative DNA damage in female dry cleaners. Mutation Res 2003;539:9–18. [PubMed: 12948810]
24. American Cancer Society: Cancer facts and figures for African Americans 2005–2006. American Cancer Society; 2006.
25. Kristal AR, Patterson RE, Glanz K, Heimendinger J, Hebert JR, Feng Z, et al. Psychosocial Correlates of Healthful Diets: Baseline Results from the Working Well Study. Prev Med 1995;24:221–228. [PubMed: 7644443]
26. Kristal AR, Curry SJ, Shattuck AL, Feng Z, Li S. A Randomized Trial of a Tailored, Self-Help Dietary Intervention: The Puget Sound Eating Patterns Study. Prev Med 2000;31:380–389. [PubMed: 11006063]
27. Ulrich C, Kristal AR, White E, Hunt JR, Durfy SJ, Potter JD. Genetic testing for cancer risk: a population survey on attitudes and intention. Community Genet 1998;1:213–222. [PubMed: 11658005]
28. Mayne ST. Antioxidant Nutrients and Chronic Disease: Use of Biomarkers of Exposure and Oxidative Stress Status in Epidemiologic Research. J Nutr 2003;133:933S–940S. [PubMed: 12612179]
29. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Am J Clin Nutr 1998;68:899–917. [PubMed: 9771869]
30. Craft N. High resolution HPLC method for the simultaneous analysis of carotenoids, retinoids, and tocopherols. FASEB J 1996:A527.(abs).

31. Craft NE, Brown ED, Smith JC. Effects of storage and handling conditions on concentrations of individual carotenoids, retinol, and tocopherol in plasma. Clin Chem 1988;34:44–48. [PubMed: 3338183]
32. Guetens G, De Boeck G, Highley M, van Oosterom AT, de Bruijn EA. Oxidative DNA damage: biological significance and methods of analysis. Crit Rev Clin Lab Sci 2002;39:331–457. [PubMed: 12385502]
33. Collins A, Dusinská M, Franklin M, Somorovská M, Petrovská H, Duthie S, et al. Comet assay in human biomonitoring studies: Reliability, validation, and applications. Environ Mol Mutagen 1997;30:139–146. [PubMed: 9329638]
34. Gedik CM, Collins A. ESCODD. Establishing the background level of base oxidation in human lymphocyte DNA: results of an interlaboratory validation study. FASEB J 2005;1:1982–1984.
35. da Costa KA, Niculescu MD, Craciunescu CN, Fischer LM, Zeisel SH. Choline deficiency increases lymphocyte apoptosis and DNA damage in humans. Am J Clin Nutr 2006;84:88–94. [PubMed: 16825685]
36. Kleinbaum, D.; Kupper, L.; Muller, K.; Nizam, A. Regression Analysis and Other Multivariable Methods. Pacific Grove, CA: Duxbury Press; 1998.
37. Krebs-Smith SM, Cook A, Subar AF, Cleveland L, Friday J. US adults' fruit and vegetable intakes, 1989 to 1991: A revised baseline for the Healthy People 2000 objective. Am J Pub Health 1995;85:1623–1629. [PubMed: 7503335]
38. Wallstrom P, Wirfalt E, Lahmann PH, Gullberg B, Janzon L, Berglund G. Serum concentrations of beta-carotene and alpha-tocopherol are associated with diet, smoking, and general and central adiposity. Am J Clin Nutr 2001;73:777–785. [PubMed: 11273853]
39. Shikany JM, Patterson RE, Agurs-Collins T, Anderson G. Antioxidant supplement use in Women's Health Initiative participants. Prev Med 2003;36:379–387. [PubMed: 12634029]
40. Park SY, Murphy SP, Martin CL, Kolonel LN. Nutrient Intake from Multivitamin/Mineral Supplements Is Similar among Users from Five Ethnic Groups: The Multiethnic Cohort Study. J Am Diet Assoc 2008;108:529–533. [PubMed: 18313435]
41. Huang, K. The pharmacology of Chinese herbs. Boca Raton, FL: CRC Press; 1993. Herbs with multiple actions: ginseng.
42. Trudeau E, Kristal AR, Li S, Patterson RE. Demographic and Psychosocial Predictors of Fruit and Vegetable Intakes Differ: Implications for Dietary Interventions. J Am Diet Assoc 1998;98:1412–1417. [PubMed: 9850109]
43. Campbell MK, Demark-Wahnefried W, Symons M, Jewell D, Makarushka C, Beatty B, et al. Fruit and vegetable consumption and prevention of cancer: the Black Churches United for Better Health project. Am J Public Health 1999;89:1390–1396. [PubMed: 10474558]
44. Ajzen I. The Theory of planned behavior. Organizational Behavior and Social Human Decision Processes 1991;50:179–211.
45. Moller P, Loft S. Oxidative DNA damage in human white blood cells in dietary antioxidant intervention studies. Am J Clin Nutr 2002;76:303–310. [PubMed: 12144999]
46. Loft S, Vistisen K, Ewertz M, Tjonneland A, Overvad K, Poulsen HE. Oxidative DNA damage estimated by 8-hydroxydeoxyguanosine excretion in humans: influence of smoking, gender and body mass index. Carcinogenesis 1992;13:2241–2247. [PubMed: 1473230]
47. Block G, Dietrich M, Norkus EP, Morrow JD, Hudes M, Caan B, et al. Factors Associated with Oxidative Stress in Human Populations. Am J Epidemiol 2002;156:274–285. [PubMed: 12142263]
48. Meagher EA, Barry OP, Burke A, Lucey MR, Lawson JA, Rokach J, et al. Alcohol-induced generation of lipid peroxidation products in humans. J Clin Invest 1999;104:805–813. [PubMed: 10491416]
49. Collins AR, Gedik CM, Olmedilla B, Southon S, Bellizzi M. Oxidative DNA damage measured in human lymphocytes: large differences between sexes and between countries, and correlations with heart disease mortality rates. FASEB J 1998;12:1397–1400. [PubMed: 9761783]
50. Collins AR. Assays for oxidative stress and antioxidant status: applications to research into the biological effectiveness of polyphenols. Am J Clin Nutr 2005;81:261S–267S. [PubMed: 15640489]

Table 1

Characteristics of Study Participants Stratified by Race (n=147)

Characteristic	African American (n=70)[1]	White (n=77)	*p* value[2]
Sex			
Male	31 (44%)	36 (47%)	
Female	39 (56%)	41 (53%)	0.76
Age			
20–28	32 (47%)	26 (34%)	
29–37	19 (27%)	26 (34%)	
38–45	18 (26%)	25 (32%)	0.26
BMI			
Normal (18.5–24.9 kg/m^2)	32 (46%)	58 (75%)	
Overweight (25.0– 29.9 kg/m^2)	38 (54%)	19 (25%)	<0.0001
Education			
Some College or less	28 (40%)	19 (25%)	
College graduate	30 (43%)	31 (40%)	
Advanced Degree	12 (17%)	27 (35%)	0.03
Income			
Less than $20,000	14 (22%)	14 (19%)	
$20,000–39,000	15 (23%)	17 (23%)	
$40,000–79,000	19 (29%)	26 (36%)	
$80,000 or more	17 (26%)	16 (22%)	0.85
Dietary Supplement Use			
None	45 (64%)	37 (48%)	
Multivitamin Only	15 (21%)	16 (21%)	
2 or More Supplements	10 (14%)	24 (31%)	0.08
Physical Activity			
Less than twice/week	40 (57%)	21 (27%)	
3–4 times per week	23 (33%)	27 (35%)	
5+ times per week	7 (10%)	29 (38%)	<0.0001
Alcohol Consumption			
Never	29 (41%)	15 (19%)	
Less than 1 per week	22 (31%)	24 (31%)	
1–6 times per week	16 (23%)	31 (40%)	
1 or more per day	3 (4%)	7 (9%)	0.01

[1] Numbers may not total 70 for African Americans and 77 for Whites due to rounding and missing data.

[2] Overall p value for African Americans compared to Whites based on chi-square tests.

Table 2

Mean Plasma Antioxidant and Oxidative DNA Damage Levels for Demographic Correlates, by Race (n=147)

	Vitamin C (μg/ml)		Vitamin E (μg/ml)		β-carotene (μg/ml)		Lutein + Zeaxanthin (μg/ml)		Lycopene (μg/ml)		Oxidative DNA Damage [1]	
	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=69)	White (n=75)
Sex												
Male	8.67	9.22	7.84	9.85	0.19	0.20	0.11	0.12	0.48	0.43	1.38	1.54
Female	8.80	8.24	7.40	10.43	0.18	0.27	0.12	0.15	0.41	0.39	1.39	1.58
Overall *p* value[2]	0.94	0.04	0.88	0.59	0.68	0.24	0.40	0.26	0.11	0.03	0.81	0.57
Age												
20–28	8.43	8.30	7.20	9.01	0.18	0.17	0.11	0.12	0.45	0.37	1.33	1.53
29–37	9.76	8.82	8.25	10.45	0.20	0.26	0.13	0.14	0.48	0.43	1.40	1.43
38–45	8.25	9.00	7.63	11.05	0.19	0.28	0.11	0.15	0.39	0.43	1.48	1.72
p for linear trend	0.79	0.26	0.63	<0.001	0.75	<0.001	0.62	0.02	0.12	0.09	0.14	0.11
BMI												
Normal (18.5–24.9 kg/m^2)	9.45	8.81	7.08	10.20	0.20	0.24	0.13	0.14	0.40	0.42	1.35	1.57
Overweight (25–29.9 kg/m^2)	8.15	8.36	8.03	10.03	0.17	0.22	0.11	0.11	0.47	0.37	1.42	1.54
Overall *p* value	0.05	0.34	0.74	0.44	0.50	0.39	0.08	0.03	0.43	0.58	0.21	0.78
Waist Circumference												
Lowest Tertile (Sex-specific)	9.46	8.92	6.30	9.42	0.20	0.23	0.11	0.14	0.39	0.40	1.34	1.53
Middle Tertile (Sex-specific)	8.72	8.81	7.90	9.83	0.23	0.24	0.14	0.14	0.51	0.40	1.37	1.59
Highest Tertile (Sex-specific)	8.20	8.07	8.36	12.23	0.14	0.24	0.10	0.13	0.42	0.44	1.44	1.58
p for linear trend	0.05	0.11	0.33	0.01	0.09	0.77	0.67	0.31	0.38	0.43	0.05	0.63
Education												
Some College or less	8.40	8.80	7.27	9.26	0.18	0.18	0.11	0.12	0.40	0.40	1.38	1.55
College graduate	9.29	8.45	7.73	9.62	0.19	0.21	0.12	0.12	0.45	0.37	1.43	1.59
Advanced Degree	8.18	8.93	8.02	11.41	0.19	0.31	0.12	0.16	0.52	0.46	1.26	1.53
p for linear trend	0.74	0.77	0.49	0.005	0.17	0.001	0.88	0.004	0.02	0.21	0.52	0.92
Income												
Less than $20,000	9.27	7.83	8.33	8.92	0.20	0.18	0.11	0.11	0.43	0.37	1.32	1.51
$20,000–39,000	7.92	8.65	6.37	9.75	0.21	0.19	0.13	0.13	0.51	0.37	1.41	1.56
$40,000–79,000	9.31	9.10	7.44	10.36	0.21	0.25	0.12	0.13	0.37	0.46	1.43	1.53
$80,000 or more	8.58	9.41	8.53	11.52	0.16	0.33	0.13	0.18	0.49	0.43	1.39	1.69
p for linear trend	0.79	0.04	0.11	0.82	0.01	0.18	0.02	0.07	0.03	0.18	0.75	0.67

[1] Oxidative DNA Damage measured as mean comet tail moment of 100 cells via the comet assay; results were unavailable for 3 participants due to missing samples.

[2] Differences between each demographic variable and the log-transformed distributions of plasma antioxidant concentration or oxidative DNA damage were assessed using t-tests, separately for total African Americans and Whites. Plasma cholesterol was included in all models of fat soluble nutrients. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

Table 3

Mean Plasma Antioxidant and Oxidative DNA Damage Levels for Behavioral Correlates, by Race (n=147)

	Vitamin C (ascorbic acid) (μg/ml)		Vitamin E (α-tocopherol) (μg/ml)		β-carotene (μg/ml)		Lutein + Zeaxanthin (μg/ml)		Lycopene (μg/ml)		Oxidative DNA Damage [1]	
	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=69)	White (n=75)
Usual Physical Activity												
<2 times per week	9.03	7.91	7.74	9.48	0.18	0.18	0.11	0.11	0.42	0.43	1.44	1.58
3–4 times per week	8.15	8.85	7.28	10.28	0.16	0.24	0.11	0.14	0.48	0.39	1.30	1.64
5+ times per week	9.06	9.14	7.78	10.54	0.28	0.27	0.16	0.15	0.43	0.41	1.32	1.48
p for linear trend	0.97	0.07	0.94	0.12	0.21	0.02	0.14	0.02	0.39	0.77	0.10	0.28
Multivitamin Use												
Yes	7.99	8.29	7.22	9.20	0.18	0.22	0.12	0.13	0.45	0.42	1.40	1.55
No	10.62	9.32	8.54	11.71	0.20	0.28	0.13	0.15	0.43	0.41	1.33	1.59
Overall *p* value[2]	0.003	0.15	0.15	<0.001	0.66	0.04	0.52	0.13	0.50	0.85	0.48	0.70
Herbal Supplement Use												
Yes	8.22	8.29	7.38	9.77	0.18	0.14	0.12	0.10	0.45	0.54	1.39	1.34
No	13.10	10.02	8.30	9.99	0.28	0.21	0.13	0.13	0.32	0.42	1.40	1.58
Overall *p* value	0.006	0.02	0.14	0.24	0.33	0.03	0.54	0.04	0.14	0.60	0.87	0.42
Passive Smoke Exposure												
Lives with a smoker	8.61	9.00	6.99	9.81	0.15	0.36	0.12	0.23	0.39	0.45	1.13	1.41
No one at home smokes	8.76	8.74	7.65	10.21	0.19	0.23	0.12	0.13	0.45	0.41	1.41	1.57
Overall *p* value	0.77	0.64	0.89	0.63	0.42	0.24	0.84	0.05	0.57	0.58	0.01	0.48
Alcohol Consumption												
Never	9.24	9.11	9.11	11.95	0.25	0.15	0.13	0.16	0.69	0.33	1.35	1.87
Less than 1 per week	7.80	8.59	8.84	9.40	0.15	0.19	0.12	0.12	0.46	0.39	1.38	1.57
1–6 times per week	9.28	8.66	6.95	10.90	0.17	0.33	0.11	0.14	0.46	0.47	1.35	1.53
1 or more per day	8.81	8.81	7.24	9.71	0.21	0.22	0.12	0.14	0.39	0.39	1.41	1.44
p for linear trend	0.87	0.72	0.33	0.72	0.37	0.08	0.46	0.98	0.05	0.14	0.85	0.02

[1] Oxidative DNA Damage measured as mean comet tail moment of 100 cells via the comet assay; results were unavailable for 3 participants due to missing samples.

[2] Differences between each demographic variable and the log-transformed distributions of plasma antioxidant concentration or oxidative DNA damage were assessed using t-tests, separately for total African Americans and Whites. Plasma cholesterol was included in all models of fat soluble nutrients. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

Table 4

Mean Plasma Antioxidant Concentrations for Psychosocial Correlates, by Race (n=147)

	Vitamin C (ascorbic acid) (μg/ml)		Vitamin E (α-tocopherol) (μg/ml)		β-carotene (μg/ml)		Lutein + Zeaxanthin (μg/ml)		Lycopene (μg/ml)	
	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=70)	White (n=77)	African American (n=69)	White (n=75)
The link between diet & cancer is:										
Strong	9.45	10.66	8.49	10.18	0.24	0.21	0.14	0.12	0.41	0.48
Moderate	9.32	8.28	7.15	10.52	0.21	0.28	0.14	0.15	0.51	0.39
Weak/None	8.08	8.56	7.52	9.73	0.15	0.20	0.10	0.13	0.41	0.41
p for linear trend	0.09	0.13	0.26	0.31	0.03	0.05	0.002	0.50	0.81	0.41
Believe antioxidants good for health?										
Yes	9.20	8.72	7.81	10.02	0.19	0.24	0.12	0.14	0.45	0.40
Not Sure/Don't Know	7.27	8.48	6.97	11.30	0.19	0.20	0.12	0.13	0.43	0.48
Overall *p* value[1]	0.01	0.89	0.86	0.43	0.40	0.68	0.99	0.98	0.91	0.11
Knowledge of FV servings										
5 or More	9.21	8.72	7.25	10.82	0.23	0.25	0.14	0.15	0.43	0.41
4 or Less	8.53	8.68	7.76	9.33	0.17	0.22	0.11	0.12	0.44	0.41
Overall *p* value	0.22	0.85	0.46	0.02	0.21	0.09	0.02	0.02	0.68	0.78
Importance of High FV diet										
Very Important	8.96	8.63	8.40	10.53	0.21	0.26	0.13	0.15	0.42	0.40
Not or Somewhat Important	8.55	8.79	7.09	9.60	0.15	0.20	0.11	0.12	0.44	0.42
Overall *p* value	0.48	0.96	0.03	0.03	0.07	0.03	0.07	0.004	0.37	0.79
Self Efficacy to Eat FV										
Very Confidant	8.38	8.91	7.76	10.09	0.20	0.24	0.12	0.14	0.47	0.39
Not or Somewhat Confidant	9.23	8.25	7.38	10.31	0.16	0.23	0.12	0.13	0.41	0.46
Overall *p* value	0.42	0.35	0.64	0.76	0.26	0.44	0.56	0.27	0.35	0.18
Able to Afford Healthy Foods?										
Yes	8.66	8.54	7.90	10.60	0.18	0.25	0.12	0.15	0.43	0.42
No or Sometimes	8.98	9.15	6.73	8.90	0.20	0.19	0.12	0.11	0.48	0.39
Overall *p* value	0.77	0.50	0.16	0.03	0.84	0.07	0.66	0.01	0.15	0.70

[1] Differences between each demographic variable and the log-transformed distributions of plasma antioxidant concentration or oxidative DNA damage were assessed using t-tests, separately for total African Americans and Whites. Plasma cholesterol was included in all models of fat soluble nutrients. $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

Table 5

Results of Regression Models[1] Relating Demographic, Behavioral, and Psychosocial Correlates with Plasma Antioxidant Concentrations (n=147)

	African American				White			
	Variable	**β coef[2]**	***p* value**	**R^2**	**Variable**	**β coef**	***p* value**	**R^2**
Vitamin C (ascorbic acid)	Herbal Supplement Use (User vs. Non-user)	0.386	0.02	0.19	Herbal Supplement Use (User vs. Non-user)	0.261	0.02	0.08
	Belief that antioxidants are good for health	0.264	0.02					
Vitamin E (α-Tocopherol)	Cholesterol (mg/dl)	0.004	<0.0001	0.25	Cholesterol (mg/dl)	0.004	<0.0001	0.47
	Belief in the importance of a diet high in FV[3]	0.142	0.03		Multivitamin Use (User vs. Non-user)	0.171	0.002	
					Age (29–37 vs. 20–28)	0.081	0.19	
					Age (38–45 vs. 20–28)	0.191	0.003	
					Belief in the importance of a diet high in FV	0.111	0.03	
β-Carotene	Cholesterol (mg/dl)	0.001	0.77	0.01	Cholesterol (mg/dl)	0.001	0.64	0.44
					Age (29–37 vs. 20–28)	0.528	<0.001	
					Age (38–45 vs. 20–28)	0.507	0.001	
					Usual physical activity (3–4x/wk vs. <2/wk)	0.453	0.005	
					Usual physical activity (5x+/wk vs. <2/wk)	0.518	0.001	
					Diet and cancer link (moderate vs. weak/no belief)	0.449	0.001	
					Diet and cancer link (strong vs. weak/no belief)	0.211	0.27	
					Lives with a smoker	−0.529	0.05	
Lutein + Zeaxanthin	Cholesterol (mg/dl)	0.004	0.77	0.18	Cholesterol (mg/dl)	0.002	0.08	0.21
	Diet and cancer link (moderate vs. weak/no belief)	0.284	0.01		Herbal Supplement Use (User vs. Non-user)	0.227	0.03	
	Diet and cancer link (strong vs. weak/no belief)	0.353	0.004		Belief in the importance of a diet high in FV	0.259	0.005	
Lycopene	Cholesterol (mg/dl)	0.004	0.02	0.25	Cholesterol (mg/dl)	0.004	0.001	0.26
	Age (29–37 vs. 20–28)	−0.088	0.40		Waist Circumference (in)	0.025	0.03	
	Age (38–45 vs. 20–28)	−0.294	0.01		Diet and cancer link (moderate vs. weak/no belief)	−0.100	0.22	
					Diet and cancer link (strong vs. weak/no belief)	0.212	0.07	

[1] Forward stepwise regression models (inclusion criterion = 0.05) were computed separately by race. Cholesterol was automatically retained in all models of fat soluble nutrients (all nutrients here, except vitamin C).

[2] β coef=Estimated regression coefficient.

[3] FV= fruits and vegetables.

Table 6

Results of Regression Model[1] Relating Relevant Demographic and Behavioral Correlates with Oxidative DNA Damage Levels (n=144)

	African American (n=69)				White (n=75)			
	Variable	**β coef[2]**	***p* value**	**R^2**	**Variable**	**β coef**	***p* value**	**R^2**
Oxidative DNA Damage	Lives with a smoker (Yes vs. No)	0.193	0.05	0.19	Lives with a smoker (Yes vs. No)	0.048	0.70	0.27
	Sex (Female vs. Male)	0.029	0.40		Sex (Female vs. Male)	0.123	0.12	
	Age (29–37 vs. 20–28)	0.034	0.63		Age (29–37 vs. 20–28)	−0.021	0.77	
	Age (38–45 vs. 20–28)	0.052	0.50		Age (38–45 vs. 20–28)	0.151	0.04	
	BMI (Overweight vs. Normal)	0.059	0.43		BMI (Overweight vs. Normal)	−0.022	0.77	
	Alcohol (<1 time/ week vs. Never)	−0.058	0.71		Alcohol (<1 time/ week vs. Never)	−0.110	0.28	
	Alcohol (1–6 times/ week vs. Never)	−0.078	0.61		Alcohol (1–6 times/ week vs. Never)	−0.242	0.02	
	Alcohol (1 or more per day vs. Never)	−0.067	0.67		Alcohol (1 or more per day vs. Never)	−0.241	0.03	
	Physical Activity (3–4 vs. <2 per week)	−0.050	0.43		Physical Activity (3–4 vs. <2 per week)	−0.030	0.68	
	Physical Activity (5+ vs. <2 per week)	−0.090	0.37		Physical Activity (5+ vs. <2 per week)	−0.089	0.21	
	Education (College grad vs. some college or less)	0.019	0.76		Education (College grad vs. some college or less)	0.093	0.18	
	Education (Adv degree vs. some college or less)	−0.115	0.20		Education (Adv degree vs. some college or less)	0.036	0.62	

[1] Multiple linear regression models were computed separately by race.

[2] β coef=Estimated regression coefficient.